EDITORIAL

Adversaries in Patient Care

ELSEWHERE IN THIS ISSUE is a timely commentary on the Medicaid experience in California in the program known as Medi-Cal. There is much to be learned from this experience and it can only be hoped that its lessons are being heeded by those responsible for designing the program of national health insurance which sooner or later will be made available to or imposed upon the people of this nation, depending upon how the law is finally written.

One of the points made by Roney in his commentary on Medi-Cal is that for any such program to work it must first be inherently workable and then there must be incentives for cooperation and a desire on the part of all concerned to make it work. The Medi-Cal program is unusual in that it was initially sponsored by organized medicine which subsequently did everything in its power to make it work, but continually found itself in an adversary position with the State administration of the program. In desperation it finally took the State itself to court in behalf of patients who had been administratively deprived of their rightful benefits under the law. The case was won but the adversary relationship continues. This has proven frustrating, costly and inefficient, and it has adversely affected patient care under the program.

There is now a generally held opinion that some form of national health insurance is both desirable and necessary. Although low in the public's assessment of national priorities, it seems destined to be bullied or jockeyed into being by the Administration and the Congress during a time of unprecedented economic inflation and unprecedented popular distrust of government. The evident absence of working health care professionals, representative of their constituencies, at the apparently substantive discussions now going on behind closed doors, reduces the likelihood of anything approaching enthusiastic support by health care professionals for whatever program emerges and presumably is enacted into law. If experience with other federal health care legislation is any criterion, the program will probably not achieve its objectives, will be costly and inefficient, and will perhaps not even be workable. All of this sets the stage for another adversary rather than cooperative relationship between those who will be administering the program after it is enacted and those who must try to deliver care to patients within it.

This does not bode well. The all-important human factors in such a human undertaking as providing for health care, such as motivation and satisfaction for those providing and receiving the care, seem not to have been taken into much, if any, account. And tragically once again it will be the patients who will inevitably bear the main brunt of it all, as was the case with Medi-Cal. It is also tragic that many of the difficulties which undoubtedly lie ahead could almost certainly be prevented even at this late date if the adversary approach, which unfortunately seems to be the order of the day, could be replaced by honest and genuine cooperation on the part of all concerned.

Unfortunately this seems most unlikely in the present climate.

—MSMW

"Inappropriate" Versus "Appropriate" Antidiuretic Hormone Secretion

IN THIS ISSUE of THE WESTERN JOURNAL OF MEDICINE Drs. Mendoza and Keller have discussed the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) and suggest that varying degrees of severity of this syndrome occur frequently in pediatric patients suffering from meningitis.

When considering SIADH several important questions arise with respect to pathogenesis, diagnosis and management in either the adult or pediatric population. What is "appropriate" and what is "inappropriate" secretion of antidiuretic hormone (ADH)? The authors emphasize that changes in plasma osmolality and volume status are two "physiologic stimuli for ADH release." Should these two circumstances then be considered the two main instances whereby "appropriate" secretion of ADH occurs? From a teleological point of view, both of these circumstances have goals which tend to preserve important

aspects of body integrity, namely constancy of osmolality and volume of body fluids. Perhaps they then deserve the classification as "appropriate" stimuli of ADH release.

What then about ADH release in response to emotions, pain, trauma and exercise? Should these be considered "inappropriate" stimuli for ADH release because they do not appear to serve any immediate function in preserving total body fluid homeostasis? What also about the impaired water excretion which may be associated with adrenal insufficiency, cardiac and hepatic failure and is probably related, at least in part, to increased release of vasopressin? With adrenal insufficiency a teleological goal could be prescribed since hypovolemia may be present, but patients with hepatic and cardiac failure generally have an excess of total body fluid. Thus, a common pathway for these varied circumstances, in which non-osmotic release of ADH occurs, does not seem to be readily apparent.

A recent series of investigations, however, suggest that autonomic neural pathways may be primarily involved in the non-osmotic release of ADH.[1-5] Specifically, parasympathetic afferent pathways from the carotid sinus and aortic arch baroreceptors have been recently shown to mediate the effects of alpha- and beta-adrenergic stimulation on vasopressin release.[4,5] Moreover, diminished parasympathetic afferent tone has been shown to mediate the effect of bilateral cervical vagotomy to stimulate vasopressin release.[3] On the background of this recent experimental evidence, it is interesting to note that many of the non-osmotic stimuli for vasopressin release might be expected to be associated with increased sympathetic tone and thus diminished parasympathetic tone. In this regard, emotional stress, pain, trauma, exercise, hypovolemia, hepatic and cardiac failure, and adrenal insufficiency might be expected to be associated with enhanced sympathetic activity.

From an evolutionary viewpoint, could then the non-osmotic release of ADH be an integral part of the autonomic neural response of the organism to a variety of stress reactions? In earlier, sea-dwelling species in which water conservation was less critical, perhaps the pressor rather than the antidiuretic action of vasopressin was more important. In the context of this hypothesis perhaps all non-osmotic release of vasopressin from the hypothalamo-neurohypophyseal tract constitutes an integral component of an "appropriate" response of the autonomic nervous system to various stress reactions. Perhaps, then, only release of ADH-like activity from tumors should be classified as "inappropriate."[6]

Turning away from these hypothetical comments about the pathogenesis of SIADH, a few perhaps more practical remarks about diagnosis and management of this entity seem appropriate. In Dr. Keller's presentation she states that by day five the patient's urine and serum osmolality became "appropriate." Again, there is danger here in the use of the word "appropriate." Serum osmolality was decreased on day five to 257 milliosmols (mOsm) per kg, a level which should not be necessary to suppress ADH release and allow maximal urinary dilution. Thus, the response of this infant was not "appropriate" on day five and evidence of continued fluid accumulation and progressive hypo-osmolality from day one to five was present. In this regard, adults who are psychogenic or compulsive water drinkers may ingest 10 to 15 liters of water daily. Despite this large fluid intake, hypo-osmolality does not develop because of the exquisite sensitivity of the osmoreceptor to suppress ADH with small (2 to 4 mOsm per kg) declines in plasma osmolality.[8] The continued presence of hypo-osmolality to a level of 257 mOsm per kg in this infant then provides evidence for continued malfunction of the osmoreceptor-ADH mechanism.

Could this infant have had a so-called "reset" osmoreceptor rather than SIADH? For example, the sensitivity of the osmoreceptor may have been preserved but may have sensed 257, rather than 270, mOsm per kg as hypo-osmolality. Alternatively, the sensitivity of the osmoreceptor may have been diminished or "sluggish" in such patients so that ADH was suppressed only in the presence of large decrements (10 to 15 mOsm per kg) in plasma osmolality. Studies are needed in such patients, whose urine is diluted only when profound hypo-osmolality is present, to distinguish between "reset" and "sluggish" osmoreceptors.

The interpretation of urinary sodium concentration and excretion in patients with SIADH has also led to some confusion. Volume expansion secondary to water retention may indeed lead to increased sodium excretion and some negative sodium balance.[6] However, balance studies have shown that the fall in plasma sodium concentration in SIADH is accounted for to a negligible degree (2 to 3 milliequivalents [mEq] per liter) by

the negative sodium balance.[9] As Dr. Mendoza indicates, hypertonic saline, therefore, is not the treatment of choice in SIADH but rather a negative water balance is the goal. In chronic cases of SIADH water restriction is the preferred method of treatment. Recently, it has been shown that even with acute, symptomatic hyponatremia, hypertonic saline need not be used. The therapeutic approach described is to rapidly increase free-water clearance and correct hyponatremia by inducing diuresis with furosemide, as the urinary electrolyte losses are concomitantly replaced by a parenteral hypertonic infusion.[10] Such an approach avoids further volume expansion in patients with SIADH who already have an excess volume of total body fluids. Moreover, as Dr. Mendoza emphasizes, in the presence of this volume expansion the hypertonic saline load will be rapidly excreted.[6]

Since volume status, and not serum sodium concentration, is the main determinant of urinary sodium concentration and excretion, the authors' statement, "when the serum sodium concentration is low, it is appropriate to have a urine sodium concentration of nearly zero, with subsequent sodium retention and correction of the hyponatremia," is misleading. Urinary sodium concentration in patients with SIADH, as in normal subjects, can reach zero to 1 mEq per liter when their intake of sodium is restricted.[9] The high urinary sodium concentration in patients with SIADH is primarily related to the fact that their sodium intake must be excreted in a small volume of urine and thus necessitates a high urine sodium concentration. It is therefore important to re-emphasize that the primary defect in SIADH is that of water retention and thus, until inhibitors of ADH are available, a negative water balance achieved either by water restriction or diuretic-induced free-water losses would seem to be the most "appropriate" means of treatment. Since the role of a negative sodium balance in the pathogenesis of the hyponatremia of SIADH is negligible, the use of hypertonic saline in the treatment would seem "inappropriate."

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The Impact of Recent Findings Concerning Vitamin D Metabolism on Clinical Medicine

PERHAPS ONE OF THE most important medical advances in the last decade is the discovery that vitamin D must be converted to a hormone or hormones before it can carry out its known functions. The only known hormone produced from vitamin D is 1,25-dihydroxyvitamin D_3 ($1,25[OH]_2D_3$) but others may follow in view of the multiple activities of vitamin D. Of great importance is the fact that $1,25(OH)_2D_3$ production is feedback regulated by both serum calcium and serum phosphorus concentrations either directly or indirectly. The serum calcium regulation is mediated by the parathyroid glands with the parathyroid hormone stimulating synthesis of $1,25(OH)_2D_3$. The actual cellular or molecular mechanism of the regulation is not understood at the present time but at least it is important to realize that serum calcium and serum phosphorus feedback controls the synthesis of a hormone ($1,25[OH]_2D_3$) whose responsibility is to elevate the concentration of these ions in the plasma. Additionally, it must be recognized that regulation of the 25-hydroxyvitamin D_3-1-hydroxylase does not take place in vitamin D deficiency.

The article by Coburn and his associates discusses the regulation of $1,25(OH)_2D_3$ production but the picture presented is not clear and it is uncertain whether these authors believe that parathyroid hormone is an important regulator, a concept firmly held by a number of investigators in the field. This is particularly an important concept in